## Pial Relations with Spinal Cord Veins Explain MRI Occult Spinal AV Shunts

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The anatomic findings (figures 1-6) demonstrate the different relations between the ventral spinal cord veins and the pial network with the leptomeninges of the cord. The ventral veins are subpial buried in the depths of the ventral fissure; the pial network is located in the subarachnoid space.

The visibility of the spinal veins therefore depends on the group of veins recruited to drain an arteriovenous shunt. The few cases reported with "occult" MRI examinations and AV shunts secondarily demonstrated by angiography (figures 7 and 8) illustrate this difference.



Figure 1 Thoracic spinal cord ventral view. 1) anterior funiculus; 2) anterior root of the Th 10 spinal nerve; 3) radiculo medullary artery; 4) anterior spinal artery. The subjacent veins are also covered by the linea splendens (arrowheads).



Figure 2 Thoracic spinal cord posterior view. The funiculi (3) as well as the veins (2) and the posterior root of the spinal nerves (1) are seen through the arachnoid tissue partially densified (arrowheads).

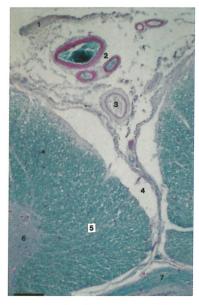


Figure 3 Horizontal section of thoracic spinal cord (luxol fast blue). 1) Linea splendens; 2) Anterior spinal artery and its branches; 3) Ventral spinal cord venous tronc; 4) Anterior medial fissure; 5) Anterior funiculus; 6) Anterior horn; 7) Spinal cord commissure (bar = 300 micrometres).

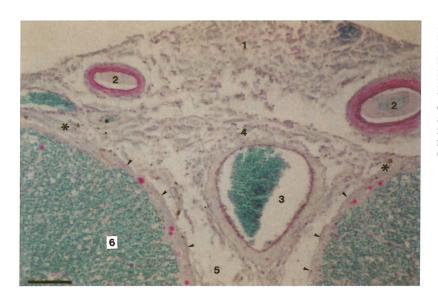


Figure 4 Horizontal section of the spinal cord at cervical level (luxol fast blue). 1) Linea splendens; 2) Unfused anterior spinal artery; 3) Anterior spinal vein covered by fasciculated pia matter. At the level of the ventral medial fissure (5) the technical staining and preparation has separated the pia glia (arrowhead). In its most anterior portion (6), the pia mater remains compact (asterisk) (bar = 200 micrometres).

Slow flow lesions draining predominantly into the ventral vein of the cord may be unseen on MRI (figure 8).

The ventral vein being in the depths of the ventral sulcus cannot be seen with myelographic type of sequences. On the contary, any lesion draining predominantly into the pial network and particularly the posterior group, are expected to be demonstrated on myelographic sequences. Normally enlarged pial veins are in fact subarachnoid and can therefore be seen without pathological significance.

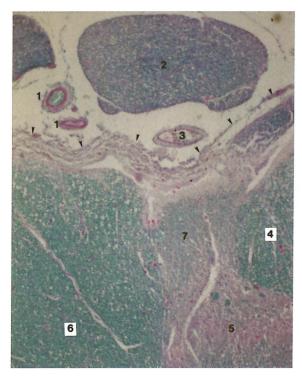


Figure 5 Horizontal section of the cord at cervical level (luxol fast blue). 1) Pial arterial vessels; 2) Posterior root of the spinal nerve; 3) Posterior venous trunk, superficial to the pia mater (arrowheads); 4) Lateral funiculus; 5) Posterior horn; 6) Posterior funiculus; 7) Posterior longitudinal fasciculus (bar = 200 micrometres).

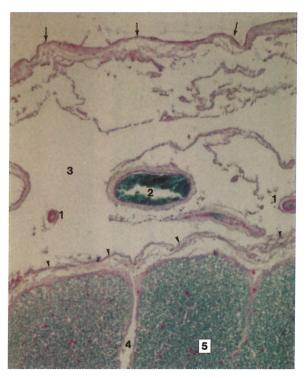


Figure 6 Horizontal section of the cord at the cervical segment. The arterioles (1) and the posterior spinal vein (2) run in the subarachnoid space (3). The arachnoid (arrows) and pia mater (arrowheads) are clearly demonstrated. (4) Medial posterior septum (5) posterior funiculus (bar = 200 micrometres).

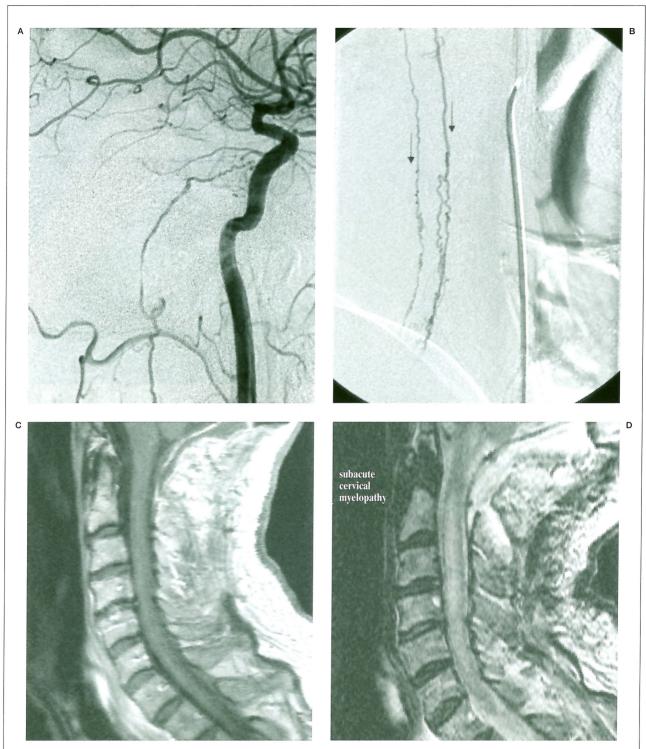
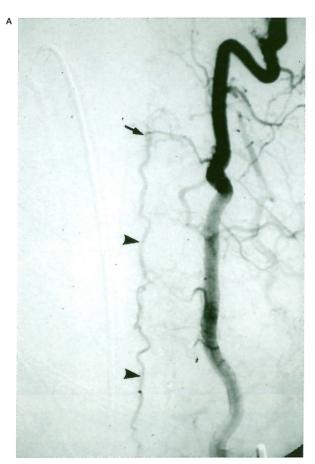
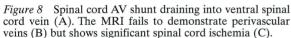


Figure 7 Dural AV shunt of the tentorium draining into the spinal cord veins (A and B). The MRI fails to demonstrate perivascular veins (C and D); note the dominant drainage into the ventral spinal cord vein.

This disposition suggests that the ischemic consequences related to ventral vein congestion in comparison to the subarachnoid congestion may very well differ. The observed absence

of haemorrhage in spinal dural arteriovenous shunt may be related to this anatomic distinction. In addition, the absence of subarachnoid haemorrhagic episodes in most spinal AV





shunts expresses an unknown property of these veins. It seems that there is no haemorrhagic venous infarction as a consequence of subarachnoid venous congestion. Instead, the subpial congestion resulting from ventral spinal cord vein involvement can lead to manifestations similar to those observed with dural or intracranial pial lesions.

The linea splendens may represent ventrally a protective barrier to the congestion provoked by a spinal dural shunt and involve only the subarachnoid network. Further observations both anatomic and hemodynamic are needed to fully understand the physiopathology of spinal AV shunts and their clinical expressions. Ignorance of the venous patterns and absence of venous analysis of spinal cord angiograms still limit discussions on spinal cord ischemia to the arterial causes.





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